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# ON

# AORTIC ANEURISM IN THE ARMY,

AND

# THE CONDITIONS ASSOCIATED WITH IT.

BY

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COMMUNICATED BY

GEO. D. POLLOCK, M.D.

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To any one perusing the literature of this subject in the text books of our schools, the idea must occur how unstable and various are the opinions advanced in regard to the causation and surroundings of these vascular lesions, and how little knowledge we possess of the disease which is placed upon a basis not open to question, and beyond the region of doubt. There is a strange discrepancy of opinion current in the military and civil segments of the medical community both in reference to ascribed external physical causations of the lesion, as well as to internal generating conditions; among the latter I especially include the influence of the syphilitic virus, an opinion firmly held by many military medical men, but more than doubted by the civil medical community at large, the

reason probably being that (in the words of the late Professor Parkes) "no analysis of cases has been made, and therefore at present its effects must be considered uncertain."

Doubtless it may be said by some that the circumstances attending military life are so special and peculiar as to make the deductions arrived at, in reference to any disease prevalent among soldiers, comparatively worthless for application to the community at large; and although this may hold good of certain limited spheres of medical and surgical science, yet I think it will be patent to all that such an objection has no validity in regard to the subject under discussion in this paper. Human nature is the same, to be impressed by general agencies, whether clothed by the uniform or under the diversified garment of civil occupation, and it may moreover be urged that the surrounding conditions and circumstances of a soldier's life are so precise and well known as compared with those of his civil brother as to render deductions on disease made from this segment of the community of much greater value and trustworthiness.

Thirty-four fatal cases of aortic aneurism are fully detailed in the pathological records of the Royal Victoria Hospital, Netley, and these form the basis of the paper. The average death age is 32 years but ranging from 26 to 42 with an average period of service of 12 years, and ranging from 4 to 21¼ years, thus embracing the entire course of military life. The average duration of the lesion is $1\frac{1}{12}$ years, varying from 3½ months to $2\frac{8}{12}$ years, but this is necessarily calculated from the time the disease became sufficiently pronounced to render the sufferer cognisant of its existence, to the date of death, and consequently is decidedly within the period from which the dilatation of vessel dates; it represents what may be termed the clinical duration of the malady. In 5 cases the aortic dilatation was multiple; in one two sacs projected from the transverse portion of the arch, one superiorly, one inferiorly; in one with two sacs from the transverse arch

posteriorly, was a third (largest and death-causing) from the descending thoracic; in two there was a thoracic and abdominal sac; and in one, with a thoracic sac were three abdominal sacs; and in two instances an aneurism of the innominate artery was conjoined with the aortic dilatation. As regards the form of the dilatation in 6 this was fusiform, viz. 5 thoracic (3 embracing ascending and transverse portion of arch, 1 transverse, 1 transverse and descending portion), and 1 abdominal; in the remainder the sacculated variety was found. In 1, the sacculated aneurism came under the category of false, the sac seated in the heart's substance and taking origin from immediately above the aortic valve; in 4 of the fusiform cases there was a false sac in connection with the original sac; in one of the sacculated cases this also existed; and in one a dissecting aneurism was situated in the walls of a sacculated abdominal aneurism. On the point of site of the sac the following table is explanatory, brought out as a percentage to exemplify relative frequency.

| | | |
|---|---|---|
| Ascending portion of arch | 26·1 per cent. | Arch of aorta 64 per cent. |
| Ascending and transverse combined [1] | 9·5 „ | |
| Transverse | 26·1 „ | |
| Transverse and descending combined [1] | 2·3 „ | |
| Descending thoracic aorta | 16·6 „ | |
| Abdominal aorta | 19· „ | |

; the thoracic lesion predominating over the abdominal a little in excess of the ratio of 5 to 1.

In 24 of the cases the condition of the heart was clearly ascertained as follows:—in 9 or 37·5 per cent. it was normal; in 11 or 45·8 per cent. (taking 10·07 oz. as the average weight of the viscus in health), it was enlarged, in 6 of the cases generally, in 5 limited to left ventricular hypertrophy; in 3 or 12·5 per cent. it was atrophied, reduced in size, but normal in structure; in 1 or 4·1 per cent. it was fattily degenerated. Hence these cases tend to show that a heart diverging from the normal standard in size or condition is no necessary asso-

[1] *i. e.*, both portions implicated in the sac.

ciate of aortic aneurism, and they also indicate that the cause of aortic aneurism cannot as a rule be linked with an over-acting viscus extra-forcibly ejecting the blood and overcoming the normal recoil of the arterial walls. The inference is rather that divergence in form and structure of the heart follows the arterial lesion, in proportion to the obstacle to the blood current and to the constitutional capacity of the system to meet the altered demands of the viscus.

Turning to the statistics of its dispersion throughout the service, we find that during the decennial period, 1863—1872, the loss by combined deaths and invaliding from aortic aneurisms alone was as follows in the three chief countries now occupied by British troops:

| | | |
|---|---|---|
| Home force (cavalry, artillery, and infantry) . | ·55 | per 1000 of strength. |
| Mediterranean garrisons (infantry, artillery, and engineers) . . . . . | ·50 | „ „ |
| India (cavalry, artillery, infantry, engineers, &c.) | ·47 | „ „ |

The home segment, with an average yearly strength of 68,760 men, during the period of ten years suffered an average annual loss from aortic aneurisms of thirty-eight men.

The component branches of the service constituting the home segment during this period gave a loss as under.

| | | |
|---|---|---|
| Cavalry (household and line) . . . . | ·53 | per 1000 of strength. |
| Artillery . . . . . | ·69 | „ „ |
| Infantry (foot guards and line regiments) . . | ·52 | „ „ |

Inspector General Lawson, taking the station of Aldershot as an exponent, concluded from the statistics of the disease during 1867–8, "that aneurism was not connected with any particular arm of the service, and even in the infantry was very irregularly distributed," for example, "out of an average of 10·2 foot corps, deaths from aneurism appeared in four of them only, while one third of the whole number of the cases of the disease was

met with in one regiment, and all apparently under the same conditions of dress, duties, &c." ('Blue Book' for 1868, p. 269.)

These figures indicate that the causes of aortic aneurism in the service are generally dispersed, not peculiar to climate, station, segment, or branch, while it is equally apparent that in the components of the respective branches of the service considerable diversity exists, and the thirty-four cases of this paper show that these causes are not connected with any special age, nor any condition of system brought about by mere length of service. What these causes are is an important problem to solve, one that it is impossible to over-estimate; and on this point the evidence furnished by the morbid anatomy and life-history of these cases seems conclusive. The deductions arrived at may be placed in the form of two propositions.

1. *That the aneurismal tumours are associated with, and preceded by, a diseased condition of the contiguous layers of the internal and middle coats of the vessel—a tissue growth terminating in degeneration—which, by impairing the elasticity and contractility of the walls, allows of their expansion and dilatation under the tension of normal arterial blood pressure, or this abnormally increased by any cause.*

This diseased state of the vessel walls comes under the nomenclature of atheroma, an extremely unsatisfactory designation, inasmuch as, taking the word in its present accepted meaning as expressive of a phase of fatty degeneration, it conveys but part of a truth, and that not the most important, and allows of the accumulation under one head ing of structural changes divergent in origin and progress.

This disease in its early stage is met with as small, elevated, translucent dots, situated immediately beneath the serous surface, irregularly scattered or linearly arranged, by coalescence assuming any shape of outline; they are due to material added.[1] They increase in thick-

[1] I find that the combined internal and middle coats of the aorta in health average $\frac{4-5}{100}$ of an inch in thickness, while in this disease I have observed a thickness of $\frac{9}{100}$ inch, due to excess of bulk of the internal coat alone from material added to it.

ness and extent, and subsequently become opaque white, or occasionally mottled red or black from blood-colouring, and from their origin in scattered foci of tissue germination, by coalescence, the inner surface of the vessel is rendered nodulated and furrowed, the furrows not uncommonly being linear and in the direction of the vessel. A vertical section through the diseased walls shows the added material as hillocks of firm tissue between the internal and middle coats, and by dissection this tissue is found mainly to be connected with the internal one (Pl. IV, figs. 1 and 2).

Under the microscope the new tissue consists of fibrous tissue cells and fibres, with very numerous nuclei in a free state (Pl. IV, fig. 3), apparently interspersed among the normal constituents; it comes under the category of growths due to localised germination of normal tissue elements, a phase of the so-called chronic inflammation, an end-arteritis. Up to this point the calibre of the vessel is unquestionably encroached upon; instead of the area being increased it is decreased from the projection within it of the more or less numerous nodular elevations (Pl. IV, fig. 1). Should the disease rest here, the vessel walls become permanently thickened, indurated, with a loss of elasticity; but as a rule the new material, following the general law of abnormal tissues, retrogresses, the node breaks up from the centre, fat globules, caseous-like particles, phosphatic crystals, and cholesterine gradually replace the fibrous tissue elements (Pl. IV, fig 6). This phase is, to the naked eye, associated with increasing opacity and softening of the patch, and then rightly comes within the strict meaning of atheroma—a reduction to a gruel-like fluid; the elevation of the inner surface of the vessel disappears, the coats reach their normal thickness or become thinner, the internal one still retaining its glistening appearance, but thrown into wrinkles by the gradually absorbed subjacent node; and so the aorta may be left with a cicatricial-like puckering of its walls and internal roughness, but no dilatation. But as a rule the formation

of abnormal material does not proceed to any great degree without implication of the inner portion of the middle coat, and consequent on the pressure from the added material and the degradation ensuing in it the walls of the vessel corresponding to the patch become decidedly impaired in structure and function. They lose their property of contracting and recoiling after distension by the blood current, the distension remains and is gradually increased, ultimately reaching a degree which brings it within the category of pronounced aneurism.

As above said the serous surface of the vessel undergoes but little if any change, retaining its translucency and glistening aspect, thrown only into folds and rugæ by the changes ensuing in the subjacent node, but occasionally it is seen to be implicated, and this in three ways: —(*a*) By the formation on its free surface of a delicate lymphy layer, generally stained by the colouring matter of the blood, and made up of extremely delicate interlacing fibres and nuclei. (*b*) By the transformation of its substance into a smooth, glistening, thin, friable, inorganic stratum, answering to the so-called ossification of arteries, and occupying the free surface of the vessel, lying on a subjacent fibroid patch, or one in process of softening. This may occasionally be seen forming a ring at the commencement of the aorta for about three quarters of an inch in extent, above which, in the continuity of the vessel, will be nodes firm or softening, gradually merging into healthy texture beyond. (*c*) It may be implicated in the disease-process, and gradually disintegrate, forming, with the subjacent changes, a sharp cut ulcer with walls and base studded with particles of degenerate tissue (Pl. IV, fig. 5). The middle coat is also occasionally seen to be involved by the production within its fibres of a distinctly circumscribed fibrous tissue nodule answering to the node formed in the voluntary muscles, and undergoing the same phases as the sub-serous thickening (Pl. IV, fig. 4, *d*). Also in the external coat may be noted the occasional presence within its loose meshes of

small, circumscribed, microscopic nodules of nuclear adenoid tissue,—lymphatic outgrowths, miliary tubercle; these being connected with each other by cordlike processes of similar material, and also with similar processes passing through the middle coat to a disintegrating internal patch. These examples suggest the normal distribution of the lymphatic tissue in the large vessels, and also outgrowths from the same under infection from the degenerating nodule towards the inside of the vessel, similar to the phases observed elsewhere under inoculation and artificial tuberculosis. Also occasionally, before any dilatation of the vessel wall has ensued, corresponding to the internal patch, is augmented vascularity of the external coat, being apparently the first stage in the natural process which ultimately forms the aneurismal sac; an intermediate stage—that of tissue production and thickening—is seen in Pl. IV, fig. 1 *d*.

As before mentioned the disease commences in separate foci ultimately more or less coalescing, and the condition of the nodules found in any given case leads to the inference either that these foci succeed each other as successive crops at different intervals or that the changes ensuing in the several masses of abnormal material are not at all uniform in time or degree. For example, in the same vessel we may find the following: (1) pouching or dilatation, general or localised, with nodulated and corrugated walls,—the most advanced of the disease already gone on to immature aneurism; (2) cicatricial-like puckering of the inner surface, with walls either of normal thickness or slightly thinned, but with no dilatation—nodes completely retrogressed and absorbed; (3) patches of thickening, opaque, soft, and friable—retrogressing; (4) nodes firm and semi-translucent, encroaching on the area of the vessel. Under these conditions it is clear either that the nodes are not all formed at the same time, or that if so formed there are great divergencies in time in the ulterior changes; the former, however, would appear to be the explanation. There is also marked

variation in the extent of vessel implicated—sometimes there is one circumscribed patch, oval in outline, and this is more often seen in the abdominal aorta; more generally the patch is irregular, more or less encircling the calibre of the vessel; at the commencement of the aorta the predominating forms are either a distinct ring, or a localised patch in one or more of the sinuses of Valsalva—under the latter phase very intense in degree generally; not uncommonly, from the site of commencement, characterised by the greater intensity and most advanced stage, the disease is seen to radiate even throughout the entire aorta, the extreme limits being marked by outlying small isolated nodules. As regards regional selection, the commencement of the aorta is at the head of the list, then the transverse portion of arch or abdominal portion just beyond the diaphragm; and in either of these or other site selected the disease may be found localised with a complete freedom of any other part of the aortic continuity.

I have dwelt thus long on the vascular disease because it seems to me to be the key to all the ulterior changes, and because a knowledge of its phases and their modifications appears to explain much that is obscure and questioned in the natural history of the aneurismal tumour. It is clear that the extent and subsequent phase of the node determine the ulterior results, and the nature and kind of the aneurismal lesion. So long as the added fibroid material remains as such, or should it be limited in extent and on retrogression be absorbed without impairment of the function of the walls, no dilatation will ensue. But should the natural function of the walls be impaired, then in the event of the disease being generally dispersed over the whole calibre and uniform in degree of degradation, we get a general dilatation, a fusiform aneurism; should the lesion be limited in extent, a mere patch, the dilatation will be limited and the aneurism sacculated, and this is not uncommonly seen combined with the former form from one patch of disease

out of a mass generally dispersed being in advance of the rest, and so inducing a sacculated aneurism projecting from a fusiform kind; should the internal coat of the vessel be implicated in the degeneration forming an open ulcer, then all the elements for the dissecting aneurism are present. It will also be apparent that an aneurism may arise from a local patch of disease, and yet no disease may be found elsewhere in the vessel; a feature clearly illustrated in the natural history of aortic nodes, and which I believe forms often the true explanation of many of those so-called examples of "aneurisms not preceded by atheromatous change," the deduction being generally made from the absence of disease elsewhere in the vessel, and the fact overlooked that the stages of thickening and atheroma are necessarily past and gone before the aneurism can ensue.

The evidence furnished by the post-mortem records of this hospital clearly shows that it is impossible in pathology to separate the aortic disease from the aneurismal lesion, the former being the precursor of the latter, and this is seen not only in those examples coming under the category of aneurism, and classified as such, but also in those cases of disease elucidated post mortem, the cause of death being otherwise than arterial lesion, but in which the aortic disease is present yet not sufficiently advanced or pronounced to give it a maximum importance in the determination of death. From the fibrous node in the internal and middle coat to the aneurism is a connected chain, which commencing as a tissue growth, abnormal in origin, leads through a fatty and caseous degeneration of the formed material to impairment of the resiliency of the arterial walls, and so under internal blood pressure to dilatation. That this degeneration of the vessel coats is no mere result of age-changes, is clear from the death ages of these cases; that a tissue growth precedes the degeneration is unquestionable; hence the important point in ætiology is to find out the causation of the growth—the conditions under which this germination of the con-

tiguous layers of the internal and middle coats of the vessel originates; for the subsequent degeneration to which it is liable, and which lays the foundation for the aneurismal dilatation, is a phase common to most abnormal growths and some normal tissues under deteriorated states of the system.

But there is another condition of the aortic walls also included under atheroma. It is met with as a more or less diffused opacity seen from the inner surface of the vessel, in the form of an irregular patch or streak; its seat being in the internal or possibly the inner layer of the middle coat. But there is no thickening of tissue conjoined with it, and no added material, and in all the examples I can find in these records it is never seen otherwise than as an opacity with no anterior or succeeding phase; I have not been able to connect it with any dilatation of the vessel, it appears a mere passive condition. Microscopically it is sometimes unquestionably fatty degeneration, and apparently of normal tissue; sometimes no fatty change can be detected, and the cause of the opalescence is far from clear. In its extent, degree, structure and ulterior results, it diverges from the nodular growth; it fairly comes under the definition of a limited opacity of the internal arterial wall, and does not appear to be followed by any deleterious result within the soldier's service—eighteen to forty years of age—and under the conditions of military life.

Thus, these two forms of aortic disease are included under atheroma: the one a passive degenerative phase apparently innocuous, the other a fibrous tissue growth with sequelæ as follow:—(*a*), it may encroach on the calibre of the aorta and produce a permanent curtailment of its area; (*b*), it may induce an indurated and inelastic condition of the walls antagonistic to the normal expansion and recoil under the blood current; (*c*), when seated in the ascending portion of the vessel it may so obstruct the onward passage of the blood as to lead to hypertrophy or dilatation of the left ventricle and death through the

damming back of the venous blood current; (*d*), by extension to the aortic valve it may effect the same end; (*e*), by softening and impairment of function of the walls it may illustrate aneurismal lesion, and (*f*), by its roughened surface, it may lead to fibrinous deposition, and through this to embolic transference to distant parts.[1]

(2nd Proposition). *That these two forms of textural derangement of the aorta are dissimilar in origin and causation; that the limited passive opacity is connected with long-standing diseases of various kinds inducing a diminished vitality of the system at large; that the structural growth is in the major number of instances associated with syphilis, and in a minor degree with rheumatism and alcoholism, as causations: hence it follows that, as the latter phase is the commencement of that pathological sequence of events under one aspect terminating as aneurism, the means for the prevention of the aneurismal tumour must be essentially directed towards the elimination of the special exciting agencies.*

Taking in the first place the thirty-four cases of aneurism, the matter stands thus:

(*a*) In constitutions undoubtedly syphilitic and nothing otherwise, 17 or 50 per cent.; (*b*) in constitutions probably syphilitic, but not beyond doubt, 5 or 14·7 per cent.; (*c*) with an acute rheumatic diathesis, 2 or 5·8 per cent.; (*d*) with excessive intemperance, but no other disease, 2 or 5·8 per cent.; (*e*) with syphilitic infection, but also rheumatism and alcoholism, 1 or 2·94 per cent.; (*f*) of no known condition of system from absence of reports, 6 or 17·64 per cent.; (*g*) with history, but no ascertainable condition of system, 1 or 2·94 per cent.

This analysis is based upon the "medical history

[1] Equally also in the vessels of the brain, both large and small, we see similar changes of thickening, dilatation, blood obstruction, thrombosis and embolism, leading to impaired and irregular function of the nerve centres, softening, and death, and under conditions of system similar to the aortic disease. The records of this hospital illustrate such cases, and indicate them as *one* form of brain disease due to the syphilitic virus.

sheet" of the man, detailing his diseases from the date of entry into the service, and the post-mortem facts. One or two of the headings require explanation. By "probably syphilitic but not beyond doubt" is meant, for example, that with a history of primary sore there are conjoined post-mortem lesions whose import might possibly be questioned, such as induration and ulceration of tonsils, or that with no history of primary sore the post-mortem lesions, although strongly suggestive of syphilis, cannot, without doubt, be classed as such; yet in these examples it must be remembered that there were no other diseased conditions with which to connect the lesions. Under (*f*) are embraced those cases whose life-records are not forthcoming, and in which the post-mortem data throw no conclusive light upon associated systemic conditions.

Hence, it is clear that 50 per cent. *at least* of these aortic aneurisms occurred in subjects with syphilitic infection, and with no other ascertainable conditions present to neutralise the deductions arrived at on the point of causation; while, on the other hand, the only other recognisable conditions present with which to connect these lesions were, the acute rheumatic diathesis and alcoholism, each represented by a percentage of 5·8.[1]

But as the aneurism is only one sequel of aortic nodulation, it is very essential, on the point of ætiology, not only to regard the surroundings of one of the pathological phases, but also the disease itself, and on this point the following details throw light.

[1] Also since this paper was written four cases of aortic aneurism have passed through my hands with brief details as under:

In one, not diagnosed during life, the history was incomplete and post-mortem incomplete. In the second there was a history of syphilitic infection, rheumatism, and alcoholism. In the third (a specimen sent to the museum) the man first suffered from continued fever and bronchitis, subsequently contracted a chancre in 1871, followed by secondary syphilis, and died of aneurism in 1875. In the fourth (also sent to museum) constitutional syphilis (roscola and iritis in 1874) formed the only admissions in the "medical history sheet," the man dying suddenly in the barrack-room from rupture of the aneurism (very small, and from the sinus Valsalva) in 1875.

Throughout the pathological records I can find 117 instances of aortic deterioration, excluding those embraced under aneurism, but including both forms of the lesion already described, and the systemic conditions with which they were associated are as follows :

| | | |
|---|---|---|
| 46·1 | per cent. | in undoubtedly syphilitic subjects. |
| 6·8 | „ | probably syphilitic, but not beyond doubt. |
| 21·3 | „ | in phthisical subjects. |
| 14·2 | „ | with no record for determining the matter. |
| 5·9 | „ | with heart disease. |
| 5·7 | „ | with various other diseases individually small. |

Here again there is a numerical preponderance with syphilitic infection ; but that which this table does not show, yet which is of immense importance in regard to aneurism, is this, that while the aortic node disease is the rule in the syphilitic diathesis, it is the exception under any other heading. For example, there are 56 cases detailed of the syphilitic virus terminating in death through special lesions, and of these 60·7 per cent. illustrate aortic nodulation and its phases, the major part of a severe type; and let this point be observed, that in about $\frac{1}{3}$rd of the node cases (*i. e.* 18 out of the 56 cases) dilatation of the vessel, either in the form of pouching or distinct sacculated projections, had actually ensued, that is to say, were in the immature stage of aneurism, and required only further development to bring them into this classification. Adding these 18 immature aneurisms, and one subsequently mentioned as due to the acute rheumatic diathesis, to the 34 already detailed, 53 cases are at hand, and of these 66 per cent. at least occurred in subjects infected with syphilis, and with no other ascertainable systemic status. But a possible objection may be raised to these deductions in this wise ;—is not the syphilitic virus so generally dispersed in the service as to considerably weaken the inference of the connection of aneurism with it in the light of effect and cause, and rather to tend to regard these lesions as merely running side by side in the same subject ? To show how far such an

objection is valid, I have collected all the cases I can find in which the non-existence of syphilitic infection may be fairly deduced both from the previous history and post-mortem data; these amount to 111 and give the following conclusions:

Five cases of aneurism or 4·5 per cent.; ·2 with acute rheumatic diathesis; 2 with alcoholism; 1 not ascertainable; all these figure in the aneurismal list. The remaining 106 non-syphilitic subjects thus exemplify the aortic disease. In 1, or ratio of ·94 per cent. the disease was severe, and had led on to dilatation, in an acute rheumatic diathesis with alcoholism; in 5 or 4·7 per cent. the disease had produced corrugation of the inner coat of the vessel but no dilatation—3 phthisical, 1 alcoholism, 1 aortic valve disease; in 29 or 27·3 per cent. the disease was slight, chiefly if not wholly to be included under the second form of this paper, mere opacity of the inner wall, 15 of these instances were associated with phthisis, and the remainder with renal affections, hepatic disease, dysentery, diabetes, scrofula, lupus, cancer in nearly equal proportions.

Considering that these aortic aneurismal tumours are associated with syphilitic infection to the extent of 66 per cent., and that nodular disease of the aorta in the service is not often met with otherwise than with it, it seems an incontrovertible deduction that syphilis is a very potent cause in the production of the vascular disease and consequently also of the aneurismal tumour. The syphilitic aortic lesion in its growth, its possible stability as a fibroid node, its degeneration, its retrogression leaving a scar-like cicatrix, its impairment of normal structures in which it occurs and in its vicinity, finds its counterpart in syphilitic bone disease, cranium for example, with its surface nodes passing on to softening, cicatricial-like loss of substance, atrophy of bone elements, &c.

It must, however, be recognised that an apparently similar nodular lesion[1] of the aorta may be produced by

[1] In saying "apparently similar," I do not wish it to be inferred that I

rheumatism, alcoholism, and possibly other conditions, such as extension of disease from the aortic valves. The influence both of the rheumatic poison and alcohol on the fibrous tissues is fully acknowledged, and it can create no wonder to find them acting as irritants upon the aortic walls inducing fibroid germination terminating in fatty degeneration. That the syphilitic virus as an exciting agency of the end-arteritis is generally dispersed in the service equally as the aneurismal disease, is clear from the statistics of the secondary lesions, and I have elsewhere shown that the aortic disease is the most common sequel to severe infection in the internal structures.[1] I am also inclined to believe that although not limited to any one period of virus evolution, it is yet not uncommonly one of the earliest produced lesions, and this feature seems to explain the comparative absence of gummata in the viscera in these cases of advanced aortic disease, the aneurism killing before the so-called tertiary lesions have had time to develop themselves.

But how then does the chest constriction from accoutrements, pack, &c., stand to this theory of the connection of syphilis and aneurism? That the aortic disease bears no relation to chest constriction and arterial obstruction, or the force of the blood expelled from an hypertrophied heart, seems to be clear from the observed post-mortem features of the disease, and the distribution of aneurism among the segments of the service. In the cases of aortic disease an enlarged heart is far from general, the disease is not at its commencement a dilatation, but on the contrary a thickening of the vessel walls,

regard the rheumatic and alcoholic lesion as identical with the syphilitic form. That there are many points of resemblance is, to my mind, clear, though it is highly probable that attention to the subject in the future will produce features differentiating the one lesion from the other. The special character of the aortic disease due to syphilis as set forth by some writers, viz. fibroid, is a feature certainly not peculiar to it, as the lesion in undoubtedly non-syphilitic subjects is often also so in the early stage. Further evidence is required to render stable these points.

[1] Blue Book, 1870, p. 384.

and an encroachment on its area; it is true that it most frequently affects the ascending aorta, but it is far from uncommon in the transverse and abdominal portions when the ascending aorta is free; and in the few instances in which an elongation and dilatation of the ascending arch could be fairly charged to an overacting heart, no disease was present. Equally in reference to the dispersion of aneurism in the service, the statistics show it to be generally distributed irrespective of climate or occupation; the infantry man in England with his pack and full accoutrements suffers no more from the disease than does the cavalry soldier; he suffers equally as much in India and the Mediterranean with a loose special climate uniform, as in England; the cavalry man in India appears worse off than in England. But while deducing that morbid anatomy, pathology, and statistics, exclude chest constrictions as a *direct* agent, in the production of aneurism in the service, it is not intended to deny its influence *indirectly*. Given the aortic disease with impairment of the aortic walls from syphilis, rheumatism, and alcoholism as a groundwork—then the obstruction to the circulation from any chest constriction and forced exertions in full marching order must tell upon the weakened vessel and cause its dilatation when possibly no such result in the diseased vessel would ensue under ordinary conditions. The accoutrements and forced exertions of the soldier stand to aneurism in the light of fostering agencies to the germs laid by syphilis, rheumatism and alcoholism.

Equally also it may be asked how this theory stands in reference to the *excess* of aneurism in the army as compared to civil life considering that in the opinion of competent observers there is no reason to suppose that the syphilitic virus is more common to the one segment of the community than to the other? But to this it may be replied that there are no reliable data at present from which accurate deductions and comparison can be instituted. There cannot be a particle of doubt that so far as

the army is concerned, we are far from having fathomed the influence of the virus, whether in the light of a producer of disability or death, and there is nothing to guide us in civil life in gauging its true import beyond an expression of opinion. It may also be said that the practice of constantly verifying the diagnosis by post-mortem examination gives an insight into the dispersion and frequency of aneurism in the service, which the civil records do not possess, and hence comparisons cannot be fairly instituted. But granting that the amount and degree of syphilis are about equal in both communities, and consequently also an equal amount of aortic disease from it, we might infer an excess of aneurism in the army from the conditions under which the soldier is placed. Aortic disease is not necessarily followed by aneurism, but so long as the disease is present, the groundwork of the aneurism is laid, and while no dilatation need ensue under ordinary arterial pressure or such as might be present under civil exertion, yet under the forced exertions, with chest constriction, of army exercises it would be difficult to understand how such a crippled tube as a degenerate and weakened aorta could resist the extra-internal pressure. Hence the groundwork being equal, an excess of aneurism in the army might be anticipated over that in civil life, from the special conditions under which the soldier is compelled to do his duty.

We may summarise the paper as under :

(1) That in the army we have a lesion of the aortic walls characterised by the presence of a fibroid growth mainly in the internal coat, which, as a rule, ultimately disintegrates ; and that this growth is connected with syphilis in a major degree, and rheumatism and alcoholism in minor degrees, as exciting agencies.

(2) That this disease of the aortic coats may retrogress without producing any marked ulterior results upon the system at large ; but if extensive or severe, as a rule, it is followed by one of three fatal phases : formation of

aneurism, implication of aortic valve, or hypertrophy, with or without dilatation of one or more of the heart's cavities.

(3) In the army there is also a lesion of the aortic walls characterised by limited opacity or fatty change of the normal textures of the internal coat; this is common to all diseases associated with prolonged general deterioration and especially lung destruction, but it does not appear, *per se*, to lead to ulterior results.

(4) That the chest constriction and temporary forced exertions to which the soldier is liable are powerful secondary causations in the production of aneurism, acting on the portion of vessel deteriorated by syphilis, rheumatism or alcoholism.

(5) That in the adoption of preventive measures against aneurism, the attention must be primarily directed against the causes of the aortic disease, notably the suppression of syphilis, and secondarily, against the conditions of dress, &c., which assist in its development.

## DESCRIPTION OF PLATE IV.

F. H. Weleh on Aortic Aneurism in the Army.

Fig. 1, natural size.—A nodulated aorta slit up, eut transversely across and straightened out, to show the relative thickness of the diseased eoats. (*a*) Internal coat extremely nodulated, encroaching on the area of the vessel; (*b*) middle eoat normal, except opposite *a*, where it is somewhat thinned; (*d*) external eoat, thiekened where the middle one is thinned.

Fig. 2, × 20 diameters.—Vertieal section through a node with part of the middle eoat displayed. (*a*) Internal eoat extensively thickened by laminated fibroid tissue, in whieh fatty or easeous degeneration has eommeneed in the form of granulcs arranged in lines; (*b*) middle eoat normal.

Fig. 3, × 500 diameters.—A fragment of the strueture of the node at an early period, the age of the partieles read from left to right; from the large nucleus, through the elongated eell, to the mature fibrous tissue strueture.

Fig. 4, × 20 diameters.—Vertieal seetion through a diseased aorta, showing the eondition of the internal and middle eoats. (*a*) Internal eoat thiekened by laminated fibrous tissue, and still more nodulated from the presence within (*b*), the middle eoat, of (*d*), a cireumseribed tumour made up of delieate fibrillæ and eommencing to soften and degenerate in the eentre.

Fig. 5, × 20 diameters.—Vertieal seetion through part of an uleer of the aortie walls. (*a*) Internal coat thiekened by fibrous laminæ; (*b*) middle coat with lines of fatty degeneration running through it; (*d*) ulcer eompletely eroding the internal eoat, and half way through the middlc one; (*e*) external coat whieh, instead of being made up of a loose meshwork, is thiekened and eondcnsed into compaet fibrous tissue, evidently for the purpose of strengthening the weakcned walls.

Fig. 6, × 500 diameters.—Elements forming a node in process of softening, in a state of atheroma. Granules, eascous-like particles, and cholesterine plates eompose the soft mass with a few phosphatie erystals, but well-formed oil globules are decidedly absent.

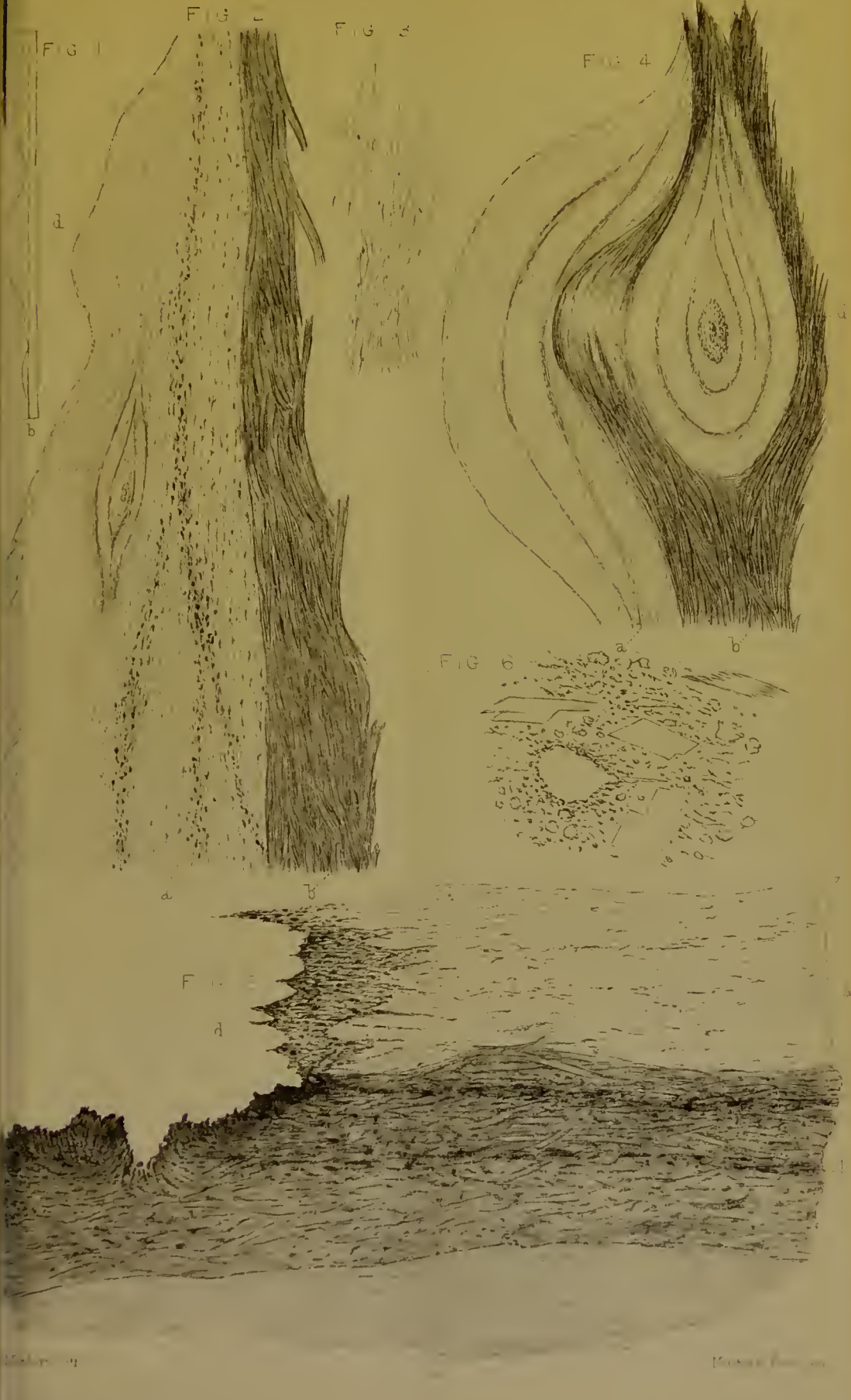
FIG 1
FIG 2
FIG 3
FIG 4
FIG 6
a
b
d